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An Association between Air Pollution and Mortality in Six U.S. Cities

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ABSTRACT

Background Recent studies have reported associations between particulate air pollution and daily mortality rates. Population-based, cross-sectional studies of metropolitan areas in the United States have also found associations between particulate air pollution and annual mortality rates, but these studies have been criticized, in part because they did not directly control for cigarette smoking and other health risks.

Methods In this prospective cohort study, we estimated the effects of air pollution on mortality, while controlling for individual risk factors. Survival analysis, including Cox proportional-hazards regression modeling, was conducted with data from a 14-to-16-year mortality follow-up of 8111 adults in six U.S. cities.

Results Mortality rates were most strongly associated with cigarette smoking. After adjusting for smoking and other risk factors, we observed statistically significant and robust associations between air pollution and mortality. The adjusted mortality-rate ratio for the most polluted of the cities as compared with the least polluted was 1.26 (95 percent confidence interval, 1.08 to 1.47). Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not with death from other causes considered together. Mortality was most strongly associated with air pollution with fine particulates, including sulfates.

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Conclusions Although the effects of other, unmeasured risk factors cannot be excluded with certainty, these results suggest that fine-particulate air pollution, or a more complex pollution mixture associated with fine particulate matter, contributes to excess mortality in certain U.S. cities.

Several cross-sectional investigations have found associations between mortality rates and particulate air pollution in U.S. metropolitan areas[1,2,3]. A recent study reported associations between infant mortality and particulate air pollution in the Czech Republic[4]. These studies have often been criticized because they did not control directly for cigarette smoking or other covariates. Recent daily time-series studies, which are likely to be free of confounding by individual characteristics, have reported associations between daily mortality rates and changes in air pollution, specifically particulate pollution, in London[5] and in several cities in the United States[6,7,8,9,10,11,12].

Particulate air pollution is a mixture of solid particles and liquid droplets that vary in size, composition, and origin. Because only very small particles can be inhaled into the lungs, U.S. national health standards for the quality of ambient air are based on the mass concentration of "inhalable particles," defined to include particles with an aerodynamic diameter of less than 10 microm[13]. Fine-particulate air pollution includes particles with an aerodynamic diameter equal to or below 2.5 microm. Whereas larger particles are derived chiefly from soil and other crustal materials, fine particles are derived primarily from the combustion of fossil fuels in transportation, manufacturing, and power generation. Fine-particulate pollution typically contains a mixture of particles including soot, acid condensates, and sulfate and nitrate particles. Fine particles are thought to pose a particularly great risk to health because they are more likely to be toxic than larger particles and can be breathed more deeply into the lungs[14].

In this study, a well-characterized cohort of adults participating in the Harvard Six Cities Study of the health effects of air pollution was followed prospectively, beginning in 1974[15]. The objective of this study was to estimate the effects of air pollution on mortality, with control for individual smoking status, sex, age, and other risk factors.

Methods

Study Population

We selected random samples of adults from six communities[15]: Watertown, Massachusetts (where study enrollment was conducted in 1974); Harriman, Tennessee, including Kingston (1975); specific census tracts of St. Louis (1975); Steubenville, Ohio (1976); Portage, Wisconsin, including Wyocena and Pardeeville (1976); and Topeka, Kansas (1977). The sample was restricted to the 8111 white subjects who were 25 through 74 years of age at enrollment, had undergone spirometric testing, and had completed a standardized questionnaire. The questionnaire included questions about age, sex, weight, height, education level, complete smoking history, occupational exposures, and medical

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history.

Informational letters and postage-paid return postcards including a question on vital status were mailed to the subjects annually. The vital status of the subjects who did not respond was determined by questioning family members, friends, or neighbors. In addition, we searched the National Death Index[16] for the years 1979 through 1989. Death certificates were obtained for 1401 of the 1430 subjects who had died (98 percent); the causes of death were coded according to the International Classification of Diseases, 9th Revision (ICD-9) by an independent certified nosologist who was blinded both to pollution levels and to the study design and objectives. The ending date of the study for each city was March or June of 1991, depending on the date of the last follow-up contact; the total duration of follow-up was 14 to 16 years (111,076 person-years).

For subjects who died, survival times were calculated by subtracting the date of enrollment from the exact date of death. For surviving participants who were not lost to follow-up, censored survival times were defined as the date of the end of the study minus the enrollment date. For those who were lost to follow-up before the period covered by our National Death Index search (i.e., before 1979), censored survival times were estimated by subtracting the enrollment date either from the date of the last follow-up contact plus six months or from the first day of the National Death Index search period (January 1, 1979), whichever came first. For those who were lost to follow-up after the National Death Index search period (i.e., after 1989), censored survival times were estimated by subtracting the enrollment date either from the date of the last follow-up contact plus six months or from the last day of the study period, whichever came first. For those who were lost to follow-up during the period covered by the National Death Index search, the censored survival times were estimated by subtracting the date of enrollment from the last date in the search period (December 31, 1989).

Air-Pollution Data

As part of the original study design, ambient (outdoor) concentrations of total suspended particulate matter, sulfur dioxide, ozone, and suspended sulfates were measured in each community at a centrally located air-monitoring station[15]. Size-selective aerosol samplers were placed at these sites in the late 1970s; data were collected for two classes of particle: fine particles (aerodynamic diameter <2.5 microm) and inhalable particles (aerodynamic diameter, <15 microm before 1984 and <10 microm starting in 1984). In the mid-1980s, supplemental 24-hour integrated sampling of aerosol acidity by the measurement of hydrogen ion concentrations[17] was conducted for approximately one year in each city. Mean pollution levels for each pollutant were calculated for periods that were consistent and comparable among the six cities.

Statistical Analysis

Life-table survival probabilities for each year of follow-up were estimated for each city, and differences between city-specific mortality rates were assessed with a log-rank test[18]. We estimated adjusted mortality-rate ratios for air pollution by simultaneously adjusting for other risk factors in

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Cox proportional-hazards regression models[18,19,20,21,22]. In these models the subjects were stratified according to sex and five-year age groups, and each sex-age group had its own base-line hazard. Each model also included indicator variables for current or former smokers, the number of pack-years of smoking (evaluated separately for current and former smokers), an indicator variable for less than a high-school education, and body-mass index (defined as the weight in kilograms divided by the square of the height in meters).

Two approaches were used to evaluate the effects of air pollution in the Cox proportional-hazards models. First, indicator variables for the city of residence were included, with Portage, Wisconsin, the city with the lowest levels of particulate air pollution, as the reference category. Adjusted mortality-rate ratios for each of the six cities were then compared graphically with the mean pollution levels in those cities. Next, adjusted mortality-rate ratios were estimated by including city-specific pollution levels directly in the Cox proportional-hazards models. Adjusted rate ratios were calculated and reported for a difference in air pollution equal to that between the city with the highest levels of air pollution and the city with the lowest levels -- that is, the adjusted rate ratios across the range of exposure for each pollutant among the six cities.

Analyses were conducted to evaluate the robustness of the models and the possibility of residual confounding. Models were estimated after the data were separated according to the subjects' smoking status, sex, and occupational exposure to dust, gases, or fumes. The effect of the inclusion of different covariates on the estimated effect of pollution was evaluated. Models were also estimated after the exclusion of subjects who had been treated for high blood pressure within 10 years of enrollment in the study and subjects who had ever been told by a doctor that they had diabetes, had glucose in their urine, or had too much glucose in their blood. We also used a variety of approaches to estimate censored survival times.

Mortality-rate ratios from the Cox proportional-hazards models (with adjustment for cigarette smoking, education, and body-mass index) were estimated separately for the following cause-of-death categories: cardiopulmonary (ICD-9 codes 400 through 440 and 485 through 496), lung cancer (162), and all others. For each cause-of-death category, data on subjects whose deaths were not in that specific category were censored at the time of death.

Results

Characteristics of the Cohort and Air-Pollution Data

The characteristics of the cohort and the values for air-pollution measures are summarized in Table 1. For all measures of air pollution except the ozone level and aerosol acidity, ambient concentrations were highest in Steubenville and lowest in Portage or Topeka. The mean acidity of the aerosol was highest in Harriman, but second-highest in Steubenville. The mean ozone concentrations were highest in Portage and Topeka. The concentrations of total particles declined during the study period, especially in Steubenville and St. Louis; the annual average concentrations of fine and sulfate

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particles varied relatively little during the study period (Figure 1). Crude mortality rates (Table 1) and survival curves (Figure 2) both show that mortality was highest in Steubenville and St. Louis and lowest in Portage and Topeka. Differences in the probability of survival among the cities were statistically significant (P<0.001).

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Table 1. Characteristics of the Study Population and Mean Air-Pollution Levels in Six Cities.

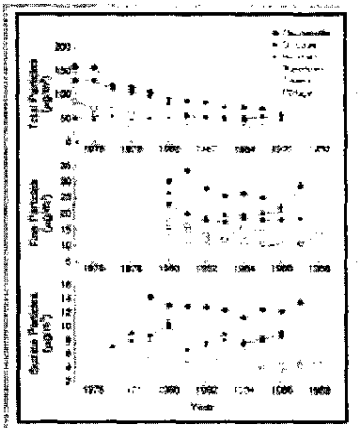

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Figure 1. Annual Average Concentrations of Total Particles, Fine Particles, and Sulfate Particles in the Six Cities.

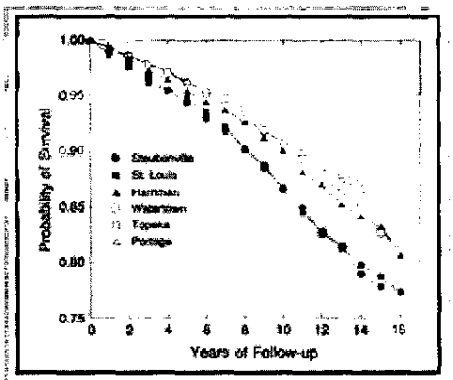

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Figure 2. Crude Probability of Survival in the Six Cities, According to Years of Follow-up.

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Adjusted Mortality Rates

On the basis of the proportional-hazards model, mortality was most strongly associated with cigarette smoking (Table 2). Increased mortality was also associated with having less than a high-school education and with increased body-mass index (the latter was especially true for women). After simultaneous adjustment for these other risk factors, the differences in mortality among the six cities remained significant.

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Table 2. Adjusted Mortality-Rate Ratios Estimated from Cox Proportional-Hazards Models.

City-specific mortality rates, adjusted for a variety of health risk factors, were associated with the average levels of air pollutants in the cities (Figure 3). The small differences in ozone levels among the cities (Table 1) limited the power of the study to detect associations between mortality and ozone levels. Mortality was more strongly associated with the levels of inhalable, fine, and sulfate particles than with the levels of total suspended particles, the sulfur dioxide levels, the nitrogen dioxide levels, or the acidity of the aerosol.

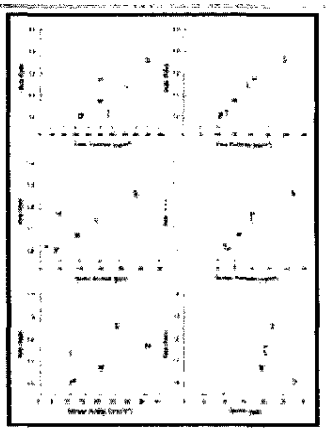

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Figure 3. Estimated Adjusted Mortality-Rate Ratios and Pollution Levels in the Six Cities.

Mean values are shown for the measures of air pollution. P denotes Portage, Wisconsin; T Topeka, Kansas; W Watertown, Massachusetts; L St. Louis; H Harriman, Tennessee; and S Steubenville, Ohio.

When the mean concentrations of each pollutant were included individually in the proportional-hazards model, we found significant associations between mortality and inhalable, fine, or sulfate particles ($P<0.005$). For a difference in the air-pollution level equal to that between the most polluted city and the least polluted city and with inhalable particles (range, 18.2 to 46.5 μg per cubic meter), fine particles (range, 11.0 to 29.6 μg per cubic meter), and sulfate particles (range, 4.8 to 12.8 μg per

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cubic meter) used as indicators of air pollution, the adjusted rate ratios were nearly equal at 1.27 (95 percent confidence interval, 1.08 to 1.48), 1.26 (95 percent confidence interval, 1.08 to 1.47), and 1.26 (95 percent confidence interval, 1.08 to 1.47), respectively.

Sensitivity

Estimates of the association between mortality and fine-particle pollution among subjects with different smoking status and among men and women (Table 3) showed only small and nonsignificant differences between subgroups. Associations with air pollution were somewhat stronger among subjects with occupational exposure to dust, gases, or fumes (Table 3). However, positive associations between mortality and air-pollution levels were observed in all subgroups defined by occupational exposure and sex, and differences among the subgroups were not statistically significant.

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Table 3. Adjusted Mortality-Rate Ratios for the Most Polluted and Least Polluted Cities Studied, According to Smoking Status, Sex, and Occupational Exposure, with Fine Particles Used as the Indicator of Air Pollution.

Although cigarette smoking and other risk factors were associated with mortality, our estimates of pollution-related mortality were not significantly affected by the inclusion or exclusion of these variables in the models (Table 4). The estimated association of air pollution and mortality was unchanged when subjects who had been treated for high blood pressure or subjects with diabetes were excluded from the analysis (Table 4). When censored survival times were recalculated as the date of the last follow-up contact minus the enrollment date, or when the analysis was restricted to data on deaths in 1979 through 1989 (the years of the National Death Index searches), no appreciable differences in the estimated association between air pollution and mortality were observed.

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Table 4. Estimated Mortality-Rate Ratios for the Most Polluted City as Compared with the Least Polluted City, with Fine Particles Used as the Indicator of Air Pollution, in Selected Models.

Causes of Death

The estimated effects of air pollution on mortality varied among causes of death (Table 5). For comparison, rate ratios were estimated for current smokers and for former smokers with approximately the average number of pack-years of smoking at enrollment (Table 5). Smoking was most strongly associated with mortality due to lung cancer, significantly associated with mortality due to cardiopulmonary disease, but not associated with mortality from all other causes. Similarly, air

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pollution was positively associated with mortality due to lung cancer and cardiopulmonary disease but not with mortality from all other causes. Only 98 deaths were coded on the death certificates as due to nonmalignant respiratory disease (ICD-9 codes 485 through 496), as compared with 646 deaths due to cardiovascular disease (codes 400 through 440). An analysis restricted to deaths from nonmalignant respiratory disease produced unstable and statistically nonsignificant estimates of the association with air pollution. When mortality from all causes was considered, or when deaths due to cardiovascular and respiratory diseases were grouped together, the effects of air pollution were consistent and the association was robust.

View this table: [in this window] [in a new window] **Table 5.** Adjusted Mortality-Rate Ratios for Current and Former Cigarette Smokers and for the Most Polluted City as Compared with the Least Polluted, According to Cause of Death.

Discussion

In this prospective cohort study, the mortality rate, adjusted for other health risk factors, was associated with the level of air pollution. Mortality was more strongly associated with the levels of fine, inhalable, and sulfate particles than with the levels of total particulate pollution, aerosol acidity, sulfur dioxide, or nitrogen dioxide. As with all other epidemiologic studies, it is possible that the observed association was due to confounding -- that is, that it resulted from a risk factor that was correlated with both exposure and mortality. Potential confounders of the effects of air pollution include cigarette smoking and occupational exposure to pollutants. In our study, however, the association of air pollution with mortality was observed even after we directly controlled for individual differences in other risk factors, including age, sex, cigarette smoking, education level, body-mass index, and occupational exposure.

The estimated effect of air pollution on mortality was not altered by the inclusion or exclusion of indicator variables for other risk factors in our models. Analyses were conducted for subgroups defined according to sex, smoking status, and occupational exposure. Although the effects of pollution were somewhat stronger among subjects occupationally exposed to dust, gases, or fumes, positive associations between mortality and air pollution were observed among all the smoking-status, occupational-exposure, and sex groups, and the differences among these subgroups were not statistically significant. The estimated association of pollution and mortality remained essentially unchanged when subjects who had been treated for high blood pressure or who had diabetes were excluded from the analysis.

In our analysis, the mortality-rate ratios have been expressed in terms of the range of exposure to air pollutants in the six cities. When the range of exposure was used, the estimated relative rate ratios for inhalable, fine, and sulfate particles were nearly equal at 1.27 (95 percent confidence interval, 1.08 to 1.48), 1.26 (95 percent confidence interval, 1.08 to 1.47), and 1.26 (95 percent confidence interval,

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1.08 to 1.47), respectively. Because the six cities were selected as representative of the range of particulate air pollution in the United States, these rate ratios roughly represent the relative risk associated with that range.

In this study, exposure to air pollution was estimated by monitoring outdoor air pollution at a central site in each of the six cities. Long-term transport and large-scale mixing of combustion products play a large part in establishing the levels of sulfate and fine-particulate air pollution. Therefore, concentrations of sulfates and fine particles are relatively uniform within each of these communities[23]. Furthermore, sulfate and fine-particulate air pollution penetrates indoors, resulting in strong correlations between indoor and outdoor concentrations[24,25,26]. Thus, measurements of the outdoor concentrations of sulfate and fine particles may be better indicators of individual exposure than the other pollutants we considered.

The associations observed in this study between air pollution and mortality are consistent with associations observed in recent time-series studies, including studies from three of these six cities[5,6,7,8,9,10,11,12]. Because the daily time-series studies evaluated only the effect of short-term changes in pollution levels, whereas our study evaluated associations with long-term exposure (including recurring episodes of relatively high pollution), quantitative comparisons with these investigations are difficult to make. Nevertheless, as was found in the time-series studies, particulate air pollution was associated with death due to cardiopulmonary causes. In our study, in which we evaluated the effects of long-term exposure, lung cancer was associated with particulate air pollution; such an association with lung cancer was not observed in the daily time-series studies. Little or no association with other causes of death was evident in our study or the time-series studies. The small number of reported deaths due to nonmalignant respiratory disease and the potential for misclassification of primary causes inherent in the use of death-certificate data limited our ability to evaluate cause-specific mortality in more detail.

The pollution concentrations used in our analysis represent only exposures monitored during the study period. Increased mortality, however, may reflect the cumulative burden of a lifetime of exposure. Concentrations of total particles clearly declined during the study period (especially in Steubenville and St. Louis), whereas concentrations of fine particles and sulfate particles were relatively stable. Given the lack of data on pollution levels before the study period and in view of the fact that the relative ranking of the cities in terms of air-pollution levels did not change during the study period, it is not possible to differentiate the influences of historical exposure from those of recent exposure. The observed association between mortality and mean exposure to fine-particulate and sulfate air pollution during the study period may also partially reflect exposure to air pollution before the study period.

The strength of the observed association between air pollution and mortality is confirmed by previous observations of associations between particulate air pollution and other health end points. Elevated levels of particulate air pollution have been associated with declines in lung function or with

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increases in respiratory symptoms such as cough, shortness of breath, wheezing, and asthma attacks[27,28,29,30,31,32,33,34,35,36]. Other studies have found associations between particulate air pollution and rates of hospitalization,[37,38,39,40,41] chronic obstructive pulmonary disease,[42] and restricted activity due to illness[43,44].

A large and growing body of literature documents the adverse health effects associated with particulate air pollution. Although the effects of unmeasured risk factors cannot be controlled for, in this prospective cohort study we observed significant effects of air pollution on mortality even when we controlled for sex, age, smoking status, education level, and occupational exposure to dust, gases, and fumes. The compatibility of the effects of air pollution on mortality in this study with those observed in population-based cross-sectional studies and daily time-series studies provides further evidence for the conclusion that exposure to air pollution contributes to excess mortality. This study, therefore, provides additional impetus to the development of strategies to reduce urban air pollution.

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Source Information

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References

1. Lave LB, Seskin EP. Air pollution and human health. Baltimore: Johns Hopkins University Press, 1977.
2. Evans JS, Tosteson T, Kinney PL. Cross-sectional mortality studies and air pollution risk assessment. Environ Int 1984;10:55-83.
3. Ozkaynak H, Thurston GD. Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal 1987;7:449-461.[Medline]
4. Bobak M, Leon DA. Air pollution and infant mortality in the Czech Republic, 1986-88. Lancet 1992;340:1010-1014.[Medline]
5. Schwartz J, Marcus A. Mortality and air pollution in London: a time series analysis. Am J Epidemiol 1990;131:185-194.[Abstract]
6. Schwartz J, Dockery DW. Increased mortality in Philadelphia associated with daily air pollution concentrations. Am Rev Respir Dis 1992;145:600-604.[Medline]
7. Kinney PL, Ozkaynak H. Associations of daily mortality and air pollution in Los Angeles County. Environ Res 1991;54:99-120.[Medline]

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8. Dockery DW, Schwartz J, Spengler JD. Air pollution and daily mortality: associations with particulates and acid aerosols. Environ Res 1992;59:362-373.[Medline]
9. Schwartz J. Particulate air pollution and daily mortality in Detroit. Environ Res 1991;56:204-213.[Medline]
10. Fairley D. The relationship of daily mortality to suspended particulates in Santa Clara County, 1980-1986. Environ Health Perspect 1990;89:159-168.[Medline]
11. Pope CA III, Schwartz J, Ransom MR. Daily mortality and PM_{10} pollution in Utah Valley. Arch Environ Health 1992;47:211-217.[Medline]
12. Schwartz J, Dockery DW. Particulate air pollution and daily mortality in Steubenville, Ohio. Am J Epidemiol 1992;135:12-19.[Abstract]
13. Environmental Protection Agency. Revisions to the National Ambient Air Quality Standards for particulate matter. Fed Regist 1987;52:24634-24669.
14. Miller FJ, Gardner DE, Graham JA, Lee RE Jr, Wilson WE, Bachmann JD. Size considerations for establishing a standard for inhalable particles. J Air Poll Control Assoc 1979;29:610-5.
15. Ferris BG Jr, Speizer FE, Spengler JD, et al. Effects of sulfur oxides and respirable particles on human health: methodology and demography of populations in study. Am Rev Respir Dis 1979;120:767-779.[Medline]
16. Stampfer MJ, Willett WC, Speizer FE, et al. Test of the National Death Index. Am J Epidemiol 1984;119:837-839.[Medline]
17. Koutrakis P, Wolfson JM, Spengler JD. An improved method for measuring aerosol strong acidity: results from a nine-month study in St. Louis, Missouri and Kingston, Tennessee. Atmos Environ 1988;22:157-162.
18. Cox DR, Oakes D. Analysis of survival data. London: Chapman & Hall, 1984.
19. Fleming TR, Harrington DP. Counting processes and survival analysis. New York: John Wiley, 1991.
20. SAS/STAT user's guide, version 6. 4th ed. Cary, N.C.: SAS Institute, 1989.
21. Cox DR. Regression models and life-tables. J R Stat Soc [B] 1972;34:187-220.
22. SAS technical report P-217, SAS/STAT software: the PHREG procedure, version 6. Cary, N.C.: SAS Institute, 1991.
23. Wilson R, Colome SD, Spengler JD, Wilson DG. Health effects of fossil fuel burning: assessment and mitigation. Cambridge, Mass.: Ballinger, 1980.
24. Dockery DW, Spengler JD. Indoor-outdoor relationships of respirable sulfates and particles. Atmos Environ 1981;15:335-343.
25. Suh HH, Spengler JD, Koutrakis P. Personal exposures to acid aerosols and ammonia. Environ Sci Tech 1992;26:2507-17.
26. Wallace LA, Pellizzari E, Sheldon L, et al. The TEAM study of inhalable particles (PM_{10}): study design, sampler performance, and preliminary results. Presented at the Annual Meeting of the Air & Waste Management Association, Vancouver, British Columbia, June 16-21, 1991. (Paper 91-171.3.)
27. Braun-Fahrlander C, Ackermann-Liebrich U, Schwartz J, Gnehm HP, Rutishauser M, Wanner HU. Air pollution and respiratory symptoms in preschool children. Am Rev Respir Dis 1992;145:42-47.[Medline]
28. Dockery DW, Ware JH, Ferris BG Jr, Speizer FE, Cook NR, Herman SM. Change in pulmonary function in children associated with air pollution episodes. J Air Pollut Control Assoc 1982;32:937-942.[Medline]
29. Ostro BD, Lipsett MJ, Wiener MB, Selner JC. Asthmatic responses to airborne acid aerosols. Am J Public Health 1991;81:694-702.[Medline]
30. Pope CA III, Dockery DW. Acute health effects of PM_{10} pollution on symptomatic and asymptomatic children. Am Rev Respir Dis 1992;145:1123-1128.[Medline]

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31. Pope CA III, Dockery DW, Spengler JD, Raizenne ME. Respiratory health and PM_{10} pollution: a daily time series analysis. Am Rev Respir Dis 1991;144:668-674.[Medline]
32. Chestnut LG, Schwartz J, Savitz DA, Burchfiel CM. Pulmonary function and ambient particulate matter: epidemiological evidence from NHANES I. Arch Environ Health 1991;46:135-144.[Medline]
33. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 1989;139:587-594.[Medline]
34. Schwartz J. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. Environ Res 1989;50:309-321.[Medline]
35. Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 1986;133:834-842.[Medline]
36. Xu XP, Dockery DW, Wang LH. Effects of air pollution on adult pulmonary function. Arch Environ Health 1991;46:198-206.[Medline]
37. Bates DV, Sizto R. Air pollution and hospital admissions in southern Ontario: the acid summer haze effect. Environ Res 1987;43:317-331.[Medline]
38. Bates DV, Baker-Anderson M, Sizto R. Asthma attack periodicity: a study of hospital emergency visits in Vancouver. Environ Res 1990;51:51-70.[Medline]
39. Ponka A. Asthma and low level air pollution in Helsinki. Arch Environ Health 1991;46:262-270.[Medline]
40. Pope CA III. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. Am J Public Health 1989;79:623-628.[Medline]
41. Pope CA III. Respiratory hospital admissions associated with PM_{10} pollution in Utah, Salt Lake, and Cache Valleys. Arch Environ Health 1991;46:90-97.[Medline]
42. Euler GL, Abbey DE, Magie AR, Hodgkin JE. Chronic obstructive pulmonary disease symptom effects of long-term cumulative exposure to ambient levels of total suspended particulates and sulfur dioxide in California Seventh-Day Adventist residents. Arch Environ Health 1987;42:213-222.[Medline]
43. Ostro BD. Associations between morbidity and alternative measures of particulate matter. Risk Anal 1990;10:421-427.[Medline]
44. Ransom MR, Pope CA III. Elementary school absences and PM_{10} pollution in Utah Valley. Environ Res 1992;58:204-219.[Medline]

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Matter. *Science* 277: 116-119 [Abstract] [Full Text]
- Bates, D. V (1996). Air pollution: time for more clean air legislation?. *BMJ* 312: 649-650 [Full Text]
- Anderson, H R., de Leon, A. P., Bland, J M., Bower, J. S, Strachan, D. P (1996). Air pollution and daily mortality in London: 1987-92. *BMJ* 312: 665-669 [Abstract] [Full Text]
- Jones, R. R. (1996). Air pollution related to transport. *BMJ* 312: 1605c-1606 [Full Text]
- Tsuda, A., Otani, Y., Butler, J. P. (1999). Acinar flow irreversibility caused by perturbations in reversible alveolar wall motion. *J. Appl. Physiol.* 86: 977-984 [Abstract] [Full Text]
- SALVI, S., BLOMBERG, A., RUDELL, B., KELLY, F., SANDSTRÖM, T., HOLGATE, S. T., FREW, A. (1999). Acute Inflammatory Responses in the Airways and Peripheral Blood After Short-Term Exposure to Diesel Exhaust in Healthy Human Volunteers. *Am J Respir Crit Care Med* 159: 702-709 [Abstract] [Full Text]
- NAEHER, L. P., HOLFORD, T. R., BECKETT, W. S., BELANGER, K., TRICHE, E. W., BRACKEN, M. B., LEADERER, B. P. (1999). Healthy Women's PEF Variations with Ambient Summer Concentrations of PM10, PM2.5, SO42-, H+, and O3. *Am J Respir Crit Care Med* 160: 117-125 [Abstract] [Full Text]
- MacNee, W., Donaldson, K. (2000). Exacerbations of COPD : Environmental Mechanisms. *Chest* 117: 390S-397 [Abstract] [Full Text]
- Su, W.-Y., Jaskot, R. H., Richards, J., Abramson, S. R., Woessner, J. F. Jr., Yu, W.-H., Dreher, K. L. (2000). Induction of pulmonary matrilysin expression by combustion and ambient air particles. *Am. J. Physiol.* 279: 152L-160 [Abstract] [Full Text]
- Mukae, H., Hogg, J. C., English, D., Vincent, R., van Eeden, S. F. (2000). Phagocytosis of particulate air pollutants by human alveolar macrophages stimulates the bone marrow. *Am. J. Physiol.* 279: 924L-931 [Abstract] [Full Text]
- Finlayson-Pitts, B. J., Pitts Jr., J. N. (1997). Tropospheric Air Pollution: Ozone, Airborne Toxics, Polycyclic Aromatic Hydrocarbons, and Particles. *Science* 276: 1045-1051 [Abstract] [Full Text]
- ZEMP, E., ELSASSER, S., SCHINDLER, C., KUNZLI, N., PERRUCHOUD, A. P., DOMENIGHETTI, G., MEDICI, T., ACKERMANN-LIEBRICH, U., LEUENBERGER, P., MONN, C., BOLOGNINI, G., BONGARD, J.-P., BRANDLI, O., KARRER, W., KELLER, R., SCHONI, M. H., TSCHOPP, J.-M., VILLIGER, B., ZELLWEGER, J.-P., the SAPALDIA Team, (1999). Long-Term Ambient Air Pollution and Respiratory Symptoms in Adults (SAPALDIA Study). *Am J Respir Crit Care Med* 159: 1257-1266 [Abstract] [Full Text]
- TAN, W. C., QIU, D., LIAM, B. L., NG, T. P., LEE, S. H., van EEDEN, S. F., D'YACHKOVA, Y., HOGG, J. C. (2000). The Human Bone Marrow Response to Acute Air Pollution Caused by Forest Fires. *Am J Respir Crit Care Med* 161: 1213-1217 [Abstract] [Full Text]
- Moolgavkar, S. H., Dockery, D. W., Pope, C. A. (1994). Air Pollution and Mortality. *N Engl J Med* 330: 1237-1238 [Full Text]
- Schenker, M. (1993). Air Pollution and Mortality. *N Engl J Med* 329: 1807-1808 [Full Text]
- TORRES, A., UTELL, M. J., MOROW, P. E., VOTER, K. Z., WHITIN, J. C., COX, C., JOHN LOONEY, R., SPEERS, D. M., TSAI, Y., FRAMPTON, M. W. (1997). Airway Inflammation in Smokers and Nonsmokers with Varying Responsiveness to Ozone. *Am J Respir Crit Care Med* 156: 728-736 [Abstract] [Full Text]
- BRUNEKREEF, B. (1999). All But Quiet on the Particulate Front. *Am J Respir Crit Care Med 159:* 354-356 [Full Text]
- POPE, C. A. III, DOCKERY, D. W., KANNER, R. E., VILLEGAS, G. M., SCHWARTZ, J. (1999). Oxygen Saturation, Pulse Rate, and Particulate Air Pollution . A Daily Time-Series

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Panel Study. *Am J Respir Crit Care Med* 159: 365-372 [Abstract] [Full Text]

- ABBEY, D. E., NISHINO, N., MCDONNELL, W. F., BURCHETTE, R. J., KNUTSEN, S. F., LAWRENCE BEESON, W., YANG, J. X. (1999). Long-Term Inhalable Particles and Other Air Pollutants Related to Mortality in Nonsmokers. *Am J Respir Crit Care Med* 159: 373-382 [Abstract] [Full Text]
- MAYER, A. S., STOLLER, J. K., BARTELSON, B. B., JAMES RUTTENBER, A., SANDHAUS, R. A., NEWMAN, L. S. (2000). Occupational Exposure Risks in Individuals with PI*Z alpha 1-Antitrypsin Deficiency. *Am J Respir Crit Care Med* 162: 553-558 [Abstract] [Full Text]
- Donaldson, K, Stone, V, Clouter, A, Renwick, L, MacNee, W (2001). Ultrafine particles. *Occup Environ Med* 58: 211-216 [Full Text]
- Kumagai, Y., Hayashi, T., Miyauchi, T., Endo, A., Iguchi, A., Kiriya-Sakai, M., Sakai, S., Yuki, K., Kikushima, M., Shimojo, N. (2001). Phenanthraquinone inhibits eNOS activity and suppresses vasorelaxation. *Am. J. Physiol.* 281: R25-30 [Abstract] [Full Text]
- Takizawa, H., Ohtoshi, T., Kawasaki, S., Kohyama, T., Desaki, M., Kasama, T., Kobayashi, K., Nakahara, K., Yamamoto, K., Matsushima, K., Kudoh, S. (1999). Diesel Exhaust Particles Induce NF-{kappa}B Activation in Human Bronchial Epithelial Cells In Vitro: Importance in Cytokine Transcription. *The JI* 162: 4705-4711 [Abstract] [Full Text]
- Lipfert, F. W. (1997). Clean Air Skepticism. *Science* 278: 17-20 [Full Text]
- Oglesby, L., Kunzli, N., Monn, C., Schindler, C., Ackermann-Liebrich, U., Leuenberger, P., the SAPALDIA Team, (2000). Validity of Annoyance Scores for Estimation of Long Term Air Pollution Exposure in Epidemiologic Studies : The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA). *Am. J. Epidemiol.* 152: 75-83 [Abstract] [Full Text]
- Samet, J. M., Dominici, F., Curriero, F. C., Coursac, I., Zeger, S. L. (2000). Fine Particulate Air Pollution and Mortality in 20 U.S. Cities, 1987-1994. *N Engl J Med* 343: 1742-1749 [Abstract] [Full Text]
- Ware, J. H. (2000). Particulate Air Pollution and Mortality -- Clearing the Air. *N Engl J Med* 343: 1798-1799 [Full Text]
- MUKAE, H., VINCENT, R., QUINLAN, K., ENGLISH, D., HARDS, J., HOGG, J. C., van EEDEN, S. F. (2001). The Effect of Repeated Exposure to Particulate Air Pollution (PM10) on the Bone Marrow. *Am J Respir Crit Care Med* 163: 201-209 [Abstract] [Full Text]
- Thun, M. J., Calle, E. E., Rodriguez, C., Wingo, P. A. (2000). Epidemiological Research at the American Cancer Society. *Cancer Epidemiol Biomarkers Prev* 9: 861-868 [Full Text]
- Bouthillier, L., Vincent, R., Goegan, P., Adamson, I. Y. R., Bjarnason, S., Stewart, M., Guenette, J., Potvin, M., Kumarathasan, P. (1998). Acute Effects of Inhaled Urban Particles and Ozone : Lung Morphology, Macrophage Activity, and Plasma Endothelin-1. *Am J Pathol* 153: 1873-1884 [Abstract] [Full Text]
- Smith, K. R. (2000). Inaugural Article: National burden of disease in India from indoor air pollution. *Proc. Natl. Acad. Sci. U. S. A.* 97: 13286-13293 [Abstract] [Full Text]
- Ohtsuka, Y., Clarke, R. W., Mitzner, W., Brunson, K., Jakab, G. J., Kleeberger, S. R. (2000). Interstrain variation in murine susceptibility to inhaled acid-coated particles. *Am. J. Physiol.* 278: 469L-476 [Abstract] [Full Text]
- Alexis, N. E., Soukup, J., Nierkens, S., Becker, S. (2001). Association between airway hyperreactivity and bronchial macrophage dysfunction in individuals with mild asthma. *Am. J. Physiol.* 280: 369L-375 [Abstract] [Full Text]
- Gilmour, P. S., Rahman, I., Hayashi, S., Hogg, J. C., Donaldson, K., MacNee, W. (2001). Adenoviral E1A primes alveolar epithelial cells to PM10-induced transcription of interleukin-8. *Am. J. Physiol.* 281: L598-606 [Abstract] [Full Text]
- Wesselkamper, S. C., Prows, D. R., Biswas, P., Willeke, K., Bingham, E., Leikauf, G. D. (2000). Genetic susceptibility to irritant-induced acute lung injury in mice. *Am. J. Physiol.* 279:

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575L-582 [Abstract] [Full Text]

- Pope, C. A. III (2000). Invited Commentary: Particulate Matter-Mortality Exposure-Response Relations and Threshold. *Am. J. Epidemiol.* 152: 407-412 [Full Text]
- Kunzli, N., Medina, S., Kaiser, R., Quenel, P., Horak, F. Jr., Studnicka, M. (2001). Assessment of Deaths Attributable to Air Pollution: Should We Use Risk Estimates based on Time Series or on Cohort Studies?. *Am. J. Epidemiol.* 153: 1050-1055 [Abstract] [Full Text]
- van EEDEN, S. F., TAN, W. C., SUWA, T., MUKAE, H., TERASHIMA, T., FUJII, T., QUI, D., VINCENT, R., HOGG, J. C. (2001). Cytokines Involved in the Systemic Inflammatory Response Induced by Exposure to Particulate Matter Air Pollutants (PM10). *Am J Respir Crit Care Med* 164: 826-830 [Abstract] [Full Text]

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